

**AMENDMENTS TO THE CLAIMS**

1. (Canceled)

2. (Currently amended) Use Method according to claim 17 wherein the antagonist of the CB1 receptor is a specific antagonist of the CB1 receptor.

3 - 11. (Canceled)

12. (Currently amended) Use Method according to any of the preceding claims claim 17 wherein the CB1 receptor is selected from the group consisting of:

- a) a protein having an amino acid sequence comprising SEQ ID NO: 1 or a portion of SEQ ID NO :1, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;
- b) a protein having an amino acid sequence comprising SEQ ID NO : 2 or a portion of SEQ ID NO : 2, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;
- c) an allele of the protein having the amino acid sequence of SEQ ID NO :1 or SEQ ID NO : 2, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;
- d) a protein having the amino acid sequence of SEQ ID NO :1 with a Phenylalanine to Leucine substitution at position 200; and/or an Isoleucine to Valine substitution at position 216; and/or a Valine to Alanine substitution at position 246;
- e) a protein having the amino acid sequence of SEQ ID NO : 2 with a Phenylalanine to Leucine substitution at position 139; and/or an Isoleucine to Valine substitution at position 155; and/or a Valine to Alanine substitution at position 185; and
- f) a protein comprising the amino acid sequences of SEQ ID NO : 3, SEQ ID NO : 4, SEQ ID NO :5, SEQ ID NO : 6, SEQ ID NO : 7, SEQ ID NO : 8 and SEQ ID NO : 9 or amino acid sequences 80% homologous to these, said protein having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal.

13. (Currently amended) Use Method according to claims 1 to 11 claim 17 wherein the CB1

receptor is a protein having a homology at the amino acid level with SEQ ID NO : 1 of at least 45%, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal.

14. (Currently amended) Use Method according to the preceding claim 13 wherein the homology is at least 60%, ~~preferably 70 %, more preferably 80 %, even more preferably 90 % and more preferably 95 %.~~

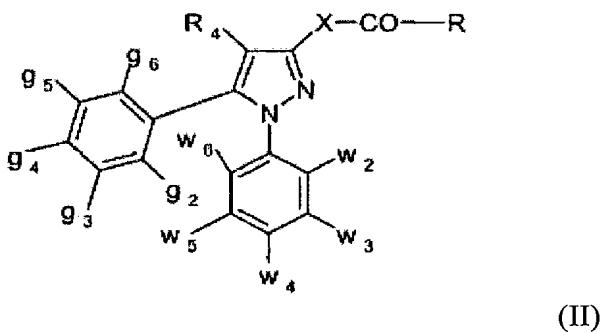
15. Currently amended) Use Method according to any of the preceding claims claim 17 wherein the daily dosage of CB1 receptor antagonist is from 0.01mg to 500mg, ~~preferably from 1 mg to 100 mg.~~

16. (Canceled)

17. (Original) A method of treatment of hepatic diseases in a mammal comprising the administration of a therapeutically effective amount of at least one CB1 receptor antagonist to a mammal in need thereof.

18. (Original) A method of treatment of hepatic diseases according to claim 17 wherein the CB1 receptor antagonist is a compound of the formula II or one of its pharmaceutically acceptable salt, in which  $g_2$ ,  $g_3$ ,  $g_4$ ,  $g_5$  and  $g_6$  and  $w_2, w_3, w_4, w_5$  and  $w_6$  are identical or different and are independently hydrogen, a chlorine or bromine atom, a (C<sub>1</sub>-C<sub>3</sub>) alkyl, a (C<sub>1</sub>-C<sub>3</sub>) alkoxy, a trifluoromethyl or a nitro group and  $g_4$  is optionally a phenyl group;  $R_4$  is hydrogen or a (C<sub>1</sub>-C<sub>3</sub>) alkyl ; X is either a direct bond or a group -(CH<sub>2</sub>)<sub>x</sub>-N(R<sub>3</sub>)-, in which R<sub>3</sub> is hydrogen or a (C<sub>1</sub>-C<sub>3</sub>) alkyl and x is zero or one; R is: a group -NR<sub>1</sub>R<sub>2</sub> in which R<sub>1</sub> and R<sub>2</sub> are independently a (C<sub>1</sub>-C<sub>6</sub>)-alkyl; an non-aromatic (C<sub>3</sub>-C<sub>15</sub>) carbocyclic radical which is optionally substituted, said substituent (s) being other than a substituted carbonyl; an amino(C<sub>1</sub>-C<sub>4</sub>) alkyl group in which the amino is optionally disubstituted by a (C<sub>1</sub>-C<sub>3</sub>) alkyl ; a cycloalkyl (C<sub>1</sub>-C<sub>3</sub>) alkyl in which the cycloalkyl is C<sub>3</sub>-C<sub>12</sub> ; a phenyl which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C<sub>1</sub>-C<sub>5</sub>) alkyl or by a (C<sub>1</sub>-C) alkoxy; a phenyl (C<sub>1</sub>-C<sub>3</sub>) alkyl ; a diphenyl (C<sub>1</sub>-C<sub>3</sub>) alkyl ; a naphthyl; an anthracenyl; a saturated 5-to 8-membered heterocyclic radical which is

unsubstituted or substituted by a (C<sub>1</sub>-C<sub>3</sub>) alkyl, by a hydroxyl or by a benzyl; a 1-adamantylmethyl; an aromatic heterocycle which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C<sub>1</sub>-C<sub>5</sub>) alkyl or by a (C<sub>1</sub>-C<sub>5</sub>) alkoxy; a (C<sub>1</sub>-C<sub>3</sub>) alkyl which is substituted by an aromatic heterocycle which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C<sub>1</sub>-C<sub>5</sub>) alkyl or by a (C<sub>1</sub>-C<sub>5</sub>) alkoxy; or else R, is hydrogen and R2 is as defined above; or else R1 and R2 form a saturated 5-to 8-membered heterocyclic radical with the nitrogen atom to which they are bonded, said heterocyclic radical being other than morpholine when w<sub>2</sub>, w<sub>3</sub>, w<sub>4</sub>, w<sub>5</sub>, w<sub>6</sub>, g<sub>2</sub>, g<sub>3</sub>, g<sub>4</sub>, g<sub>5</sub> and g<sub>6</sub> are all hydrogen; a group R<sub>2</sub> as defined above when X is -(CH<sub>2</sub>)<sub>x</sub> N(R<sub>3</sub>)- ; a group R<sub>5</sub> when X is a direct bond, R<sub>5</sub> being a (C<sub>1</sub>-C<sub>3</sub>) alkyl ; a (C<sub>3</sub>-C<sub>12</sub>) cycloalkyl which is unsubstituted or substituted by a (C<sub>1</sub>-C<sub>5</sub>) alkyl ; a phenyl(C<sub>1</sub>-C<sub>3</sub>) alkyl which is unsubstituted or substituted by a halogen or by a (C<sub>1</sub>-C<sub>5</sub>) alkyl; a cycloalkyl (C<sub>1</sub>-C<sub>3</sub>) alkyl in which the cycloalkyl is C<sub>3</sub>-C<sub>12</sub> and is unsubstituted or substituted by a(Cl-C5) alkyl ; or a 2-norbornylmethyl



19. (Original) A method of treatment of hepatic diseases according to claim 17 wherein the CB1 receptor antagonist is N-piperidono-3-pyrazolecarboxamide or one of its pharmaceutically acceptable salt.

20. (Original) A method of treatment of hepatic diseases according to claim 17 wherein the CB1 receptor antagonist is N-piperidino-5- (4-bromophenyl)-1- (2, 4-dichlorophenyl) -4-

ethylpyrazole-3-carboxamide or one of its pharmaceutically acceptable salt.

21. (Original) A method of treatment of hepatic diseases according to claim 17 wherein the CB1 receptor antagonist is N-piperidino-5- (4-chlorophenyl)-1- (2, 4-dichlorophenyl) -4-methylpyrazole-3-carboxamide or one of its pharmaceutically acceptable salt.

22. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~  
claim 17 wherein the hepatic disease is liver fibrosis.

23. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~  
claim 17 wherein the hepatic disease is alcoholic liver cirrhosis.

24. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~  
claim 17 wherein the hepatic disease is chronic viral hepatitis.

25. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~  
claim 17 wherein the hepatic disease is non-alcoholic steatohepatitis.

26. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~  
claim 17 wherein the hepatic disease is primary liver cancer.

27. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~  
claim 17 wherein the daily dosage of CB1 receptor antagonist is from 0.01mg to 500mg,  
preferably from 1mg to 100mg.